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Artificial Intelligence for Cardiovascular Disease Prevention in Resource-Limited Settings: A Review of the Literature

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Abstract

Cardiovascular prevention is effective only when risk recognition is followed by appropriate testing, prescribing, and sustained care. These steps are often disconnected in health systems with limited laboratory access, specialist availability, and longitudinal records. Artificial intelligence (AI), including machine-learning methods that learn risk patterns from large clinical datasets, may help assemble clinical and laboratory information, flag gaps in follow-up, and support decisions made by clinicians. However, most published evidence shows gains in predictive accuracy within the cohorts in which models were developed, largely in well-resourced health systems. Prospective studies have reported improvements in selected cardiovascular risk factors and care processes, but evidence that AI-supported interventions reduce first cardiovascular events in resource-limited primary prevention settings remains limited. This review of the literature synthesizes that evidence along the prevention pathway. Machine-learning studies have sometimes reported better discrimination than traditional risk equations, but performance in a development cohort does not establish reliable absolute risk in another population. Lipoprotein(a), high-sensitivity C-reactive protein (hs-CRP), and pharmacogenomic guidance can refine particular clinical and prescribing decisions; none of these demonstrate that AI-guided primary prevention improves outcomes. Randomized evidence for cardiovascular AI remains mixed and relies mostly on process or diagnostic endpoints. Community trials of fixed-dose combinations, task sharing, and digital decision support, many without AI, illustrate both effective delivery strategies and their limits. We outline a staged, clinician-supervised approach linking affordable risk assessment to treatment and follow-up and identify the validation, safety, equity, and cost questions that should precede implementation. The value of AI in prevention should be measured by improvements in patient care, not prediction accuracy alone.

Categories: Pharmacology, Cardiology, Pathology
Keywords: artificial intelligence, biomarkers, cardiovascular prevention, community health, precision pharmacotherapy, resource limited settings, risk prediction

Introduction And Background

Cardiovascular disease (CVD) continues to cause substantial preventable illness and death, although its impact and the resources available for prevention differ markedly among populations [1]. Primary prevention aims to avert a first cardiovascular event, such as myocardial infarction or stroke, in people without established disease. Risk estimation is only the beginning of prevention: a patient must also receive an intelligible recommendation, access treatment, and return for monitoring. The World Health Organization (WHO) developed region-specific cardiovascular risk charts in recognition of differences in disease patterns and the availability of laboratory measurements [2].

Conventional cardiovascular risk prediction faces four important barriers in resource-limited settings. First, access to essential laboratory investigations remains limited. The Lancet Commission on Diagnostics highlighted substantial gaps in access to basic diagnostic testing, particularly in primary healthcare settings [3]. To address this limitation, the WHO provides non-laboratory cardiovascular risk prediction charts that use body mass index instead of cholesterol measurements [2]. Second, different risk prediction tools may produce substantially different risk classifications within the same population. A cross-sectional study conducted in rural Andhra Pradesh, India, demonstrated considerable variation in the proportions of individuals classified as high risk using the Framingham, WHO, and Australian cardiovascular risk scores. The WHO score classified fewer participants as high risk compared with the other tools [4]. However, because the study did not assess subsequent cardiovascular events, these differences could not establish whether the WHO score underestimated actual cardiovascular risk.

In the same region, non-laboratory versions agreed reasonably well with laboratory versions overall but

poorly in people with diabetes [5]. Third, identifying risk does not ensure treatment. Only 9% to 12% of the high-risk adults in that rural Indian cohort were taking lipid-lowering or antihypertensive medication [4]. Across 41 low- and middle-income countries, only 8.0% of people eligible for statins for primary prevention reported using them [6], and in 44 such countries only about 10% of adults with hypertension had controlled blood pressure, with the largest single loss occurring between ever having blood pressure measured and receiving a diagnosis [7]. Fourth, inconsistent measurement can move patients between risk categories from one visit to the next, as a community trial in rural India later showed [8].

These barriers define where artificial intelligence (AI) might add value and where it cannot. In principle, AI methods could estimate risk when some inputs are missing, extract risk information from data already collected, such as electrocardiograms or chest radiographs [9], recalibrate equations to local event rates, flag implausible or incomplete entries, and predict which patients are likely to miss follow-up. AI cannot supply an unavailable medicine or replace an absent laboratory. A model that demands expensive inputs or produces a score without a feasible next step therefore adds little; the relevant test is whether an AI tool narrows one of these specific gaps better than a simpler, locally validated chart.

Established prediction equations are the appropriate starting point for assessing newer approaches. These are regression-based formulas that combine a small number of routinely measured factors, such as age, sex, blood pressure, cholesterol, diabetes, and smoking, into an estimated probability of a cardiovascular event over a fixed period, typically 10 years. The American Heart Association (AHA) PREVENT equations, for example, add kidney function and offer optional predictors; they were developed and evaluated in large US populations [10]. AI is a broader term for computational methods that learn patterns from data rather than following prespecified rules. In machine learning, the subset most relevant here, an algorithm is trained on the records of many patients whose outcomes are already known, identifies combinations of variables associated with those outcomes, and then applies them to estimate risk in new patients. Deep learning, a further subset, uses layered neural networks that can extract such patterns from electrocardiograms, images, or molecular data.

Two properties are used throughout this review to judge any risk tool. Discrimination describes how well a model ranks people who later have an event above those who do not; it is commonly summarized by the area under the receiver operating characteristic curve (AUC), where 0.5 is no better than chance, and 1.0 is perfect ranking. Calibration describes whether predicted risks match observed event rates; a tool can rank patients well yet systematically overestimate or underestimate their absolute risk. Neither property is the same as a proven change in care, and an AHA scientific statement noted the need to connect algorithm development with meaningful benefits for patients [11].

We consider prevention as a shared workflow across three specialties rather than three separate applications of AI. Pathology determines whether measurements are accurate and interpretable; pharmacology connects risk assessment with effective and safe medicines; community medicine addresses access, treatment continuity, and population reach. This perspective allows the same question to be asked at every step: does the proposed AI tool change a clinical action in a way that is affordable, reliable, and beneficial for the intended population?

The knowledge gap this review addresses is specific. Published evidence on AI for cardiovascular prevention consists mainly of improvements in discrimination within development cohorts, most drawn from well-resourced health systems [12-14]. Randomized trials of cardiovascular AI have largely measured diagnostic or process endpoints, and the certainty of evidence for clinical outcomes remains low [15]. Within the studies identified through our focused PubMed search, AI-supported interventions showed benefits for selected risk factors and aspects of clinical care. However, we did not identify a randomized trial demonstrating a reduction in first cardiovascular events through AI-supported primary prevention in resource-limited settings. Evidence on laboratory biomarkers, pharmacotherapy, and community delivery is also usually reviewed separately, so it remains unclear at which step of the prevention pathway AI could add value and which safeguards each step requires.

This review therefore had three objectives: to examine how far AI-based risk prediction improves on conventional equations when models are transported to new populations; to map evidence from pathology, pharmacology, and community medicine onto a single prevention pathway, distinguishing studies that directly tested AI from those that did not; and to propose a staged, clinician-supervised framework and the evaluation questions that should precede implementation in resource-limited settings.

Review

Review design and rationale

This article was conducted as a review of the literature using a narrative approach. The review examines the intersection of cardiovascular risk prediction, diagnostic biomarkers, personalized pharmacotherapy, and community-based prevention in resource-limited settings. Given this broad, interdisciplinary scope, the objective was to critically interpret and connect findings from prediction-model studies, biomarker research, pharmacogenetic guidelines, randomized trials, and implementation studies rather than answer a single

narrowly defined question about intervention effectiveness. Relevant publications were selected purposively according to their relevance to the review objectives, methodological quality, and clinical applicability, with additional sources identified from the reference lists of key articles. The review therefore did not employ a comprehensive, protocol-driven systematic search, formal independent screening, or study-level risk-of-bias assessment. Findings were synthesized thematically, with attention to the study design, strength of evidence, and limitations of individual sources. Reporting was informed by the six domains of the Scale for the Assessment of Narrative Review Articles (SANRA): importance of the topic, aims of the review, literature search, referencing, scientific reasoning and presentation of evidence, and relevant endpoint data [16].

Literature search and scope

We undertook a focused search of PubMed, including Medical Literature Analysis and Retrieval System Online (MEDLINE)-indexed citations, from the earliest records available in that interface to October 31, 2026; no starting publication year was specified. Searches were initially run on October 28, 2026, then broadened and repeated on October 31, 2026. Controlled vocabulary (Medical Subject Headings (MeSH)) and terms appearing in titles or abstracts were combined with AND and OR. Six searches covered cardiovascular risk prediction; biomarkers and multimodal assessment; drug treatment and adherence; screening and primary care; model validation and equity; and conventional non-AI interventions useful for understanding implementation. We did not apply electronic filters for language, population type, or study design. Appendix 1 gives the complete search strings, run dates, and retrieval totals so that the searches can be repeated.

For example, the cardiovascular risk search combined ("Cardiovascular Diseases"[MeSH Terms] OR cardiovascular[Title/Abstract] OR hypertension[Title/Abstract]) AND ("Artificial Intelligence"[MeSH Terms] OR "Machine Learning"[MeSH Terms] OR "machine learning"[Title/Abstract] OR "deep learning" [Title/Abstract]) AND ("primary prevention"[Title/Abstract] OR "risk prediction"[Title/Abstract] OR "risk stratification"[Title/Abstract] OR "Risk Assessment"[MeSH Terms]). Within-domain alternatives were joined by OR and different concepts by AND. Retrieval totals overlap across searches and do not represent individually screened or included studies. Bibliographic metadata and DOIs for cited sources were checked against publication records.

Source selection and appraisal

Relevant records and the reference lists of key articles were examined independently by two authors to identify publications relevant to the objectives of this review. Disagreements regarding the inclusion of articles were resolved through discussion and mutual consensus. We prioritized sources that met at least one of four criteria: development, external validation, or meta-analysis of AI or machine-learning models for cardiovascular risk in adults; randomized trials of AI-enabled cardiovascular tools, whatever the endpoint; biomarker, pharmacogenetic, or professional guidance directly relevant to preventive decisions; and randomized trials of conventional, team-based, or digital community interventions that clarify delivery conditions in resource-limited settings. Older trials were retained when they explain established mechanisms or service-delivery practices, and secondary-prevention studies were retained only when they clarify a mechanism or treatment effect. Digital programs that did not clearly incorporate machine learning were not treated as evidence of AI effectiveness.

Instead of numerical risk-of-bias scores, each key source was appraised qualitatively on five features that determine how far its findings apply to the review question: study design; setting (high-income or resource-limited health system); whether an AI method was directly tested; the level of the endpoint (predictive discrimination, diagnostic or process change, risk-factor control, or clinical events); and, for prediction models, whether external validation was reported. These features follow the applicability and analysis domains of the Prediction model Risk Of Bias ASsessment Tool updated for AI (PROBAST+AI) [17] and are reported for key studies in the evidence summary later in the Review. Where an included evidence synthesis had graded its own certainty or risk of bias, we report that judgment rather than re-scoring its individual studies. The searches were not used to produce a deduplicated screening dataset or pooled estimates.

Transparency, reproducibility, and bias control

Because a nonsystematic review is more susceptible to selective citation than a systematic review, we took seven steps to improve transparency and limit selection bias. First, the full Boolean search strings, databases, search dates, and retrieval counts for all six searches are provided in Appendix 1 to facilitate reproducibility. Second, the selection criteria described above guided source selection, and the rationale for including each key study is presented in the evidence summary table. Third, studies with differing findings were included, such as the null SMARThealth India trial, the overall positive PolyIran trial involving both primary and secondary prevention populations, and evidence syntheses reporting low-certainty findings. No subgroup-specific conclusion was drawn from PolyIran because of inconsistencies in the reported subgroup estimates. Fourth, key studies were identified according to whether they directly evaluated AI, preventing benefits of conventional interventions from being attributed to AI. Fifth, where evidence syntheses reported certainty or risk-of-bias assessments, we presented their findings rather than assigning independent judgments. Sixth, bibliographic details and DOIs were checked against PubMed records. Seventh, Appendix 2 identifies the sections of this review corresponding to each SANRA item.

Conventional risk prediction and the added value of AI

For primary prevention, the relevant task is not merely predicting a future event but deciding whether the estimate supports a useful intervention. Weng et al. analyzed routine records from 378,256 UK primary care patients and reported an AUC of 0.764 for their best machine-learning approach compared with 0.728 for the reference algorithm [12]. This difference suggests improved ranking in that dataset, but it does not by itself demonstrate improved treatment selection. Kakadiaris et al. also reported stronger AUC performance for a machine-learning calculator in the Multi-Ethnic Study of Atherosclerosis and examined the model in a separate European cohort [13]. Its reported performance cannot be assumed to hold at the same treatment thresholds in another health system.

Evidence syntheses reinforce the importance of context. In a 2025 meta-analysis of electronic health record prediction studies, some machine-learning methods achieved higher pooled AUCs than conventional models, but the included studies differed substantially in their populations and methods [14]. Choosing a tool for a rural clinic by comparing AUC values from unrelated cohorts would therefore be misleading. The AHA's 2026 scientific statement similarly emphasizes assessment of incremental predictive value and clinical utility against an appropriate comparator [18].

Transporting a risk equation is especially challenging when the population differs in baseline event rates, age distribution, treatment access, or completeness of follow-up. An algorithm may separate higher- from lower-risk patients reasonably well yet give inaccurate absolute probabilities. Figure *1* illustrates this calibration problem with invented values; it provides no information about individual-level discrimination. Independent assessments of the PREVENT equations underline the need to examine calibration as well as discrimination in the population in which a tool will be used [19]. For resource-limited services, the practical comparison should be with a relevant WHO chart or another locally validated method, not with model sophistication alone.

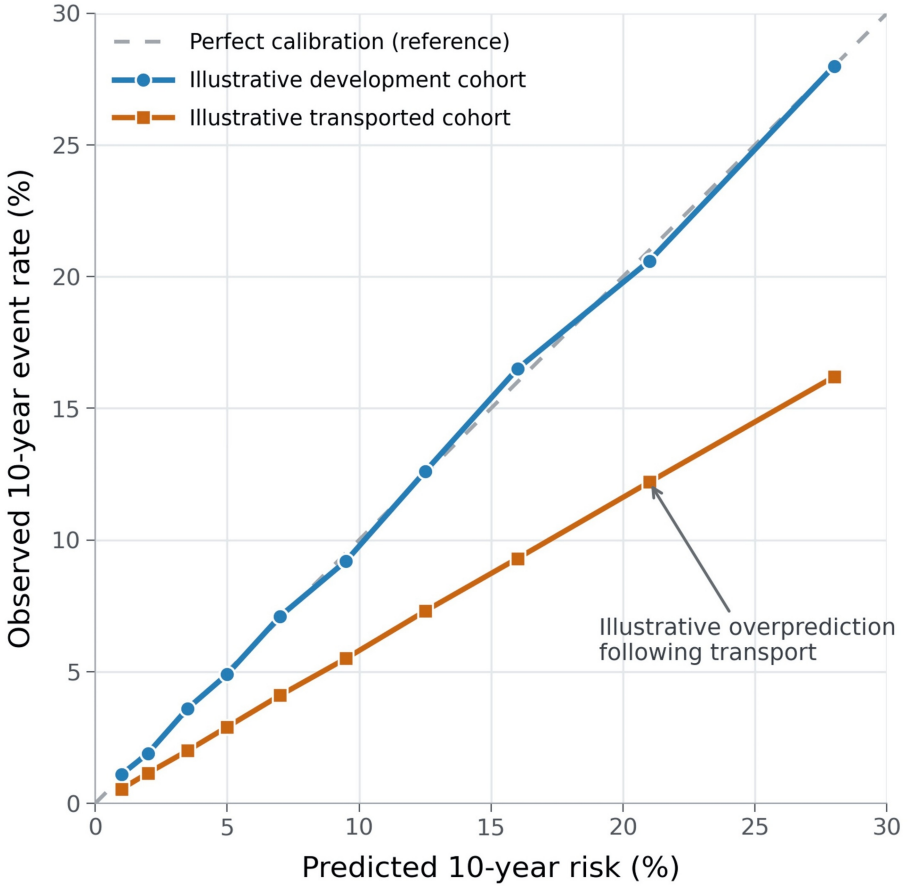


FIGURE 1: Illustrative calibration shift after transporting a cardiovascular risk model to another population

Illustrative, simulated values; not derived from patient-level data or any published cohort. Each point represents a hypothetical risk stratum. The dashed line represents perfect calibration (predicted risk equals observed event rate). The transported population has lower observed event rates than predicted, illustrating systematic overprediction and the potential need for local recalibration. Discrimination, ranking of individual patients, and clinical benefit cannot be inferred from this calibration plot.

The figure is created using Canva (Canva Pty Ltd, Sydney, NSW, Australia); Image credit: Umer Farooq and Bipin Chavda

Pathology perspective: biomarkers and multimodal risk assessment

Atherosclerosis develops through interacting lipid and inflammatory processes, including endothelial activation, recruitment of immune cells, and progressive changes within plaques [20]. These mechanisms explain why cholesterol measurements and selected inflammatory markers may contribute complementary information. Yet a plausible biological signal does not make its measurement suitable for universal screening. Laboratory reliability, turnaround time, and cost remain central constraints when preventive services are delivered at scale.

Lipoprotein(a) is largely genetically determined and may reveal cardiovascular risk that is not apparent from low-density lipoprotein cholesterol (LDL-C) measurements alone [21]. Investigators are also testing whether protein and lipid signatures add information to conventional clinical variables, using machine-learning methods to accommodate large, correlated datasets [22]. The next question is whether such information changes decisions enough to justify the assay. Preanalytical variation, between-laboratory comparability, missing values, and cost could eliminate any theoretical advantage in a low-resource clinic.

The interpretation of high-sensitivity C-reactive protein (hs-CRP) illustrates why biological and AI evidence must remain distinct. In JUPITER, rosuvastatin reduced vascular events among participants selected for elevated hs-CRP and relatively low LDL-C, without any AI-based prescribing algorithm [23]. CANTOS evaluated anti-inflammatory treatment in patients with a history of myocardial infarction and therefore informs secondary prevention rather than first-event screening [24]. These trials support specific biological or treatment conclusions, not the claim that machine learning produces superior prescribing outcomes.

Images may also yield risk-related signals without requiring an entirely new examination. Weiss et al. evaluated deep learning applied to existing chest radiographs as a possible way to estimate cardiovascular risk when conventional calculator inputs were unavailable [9]. Multimodal studies have also explored combining molecular and clinical data in a single model [25]. Neither strategy should be routinely added to resource-limited prevention until local studies show that the information improves risk calibration, alters appropriate treatment, and offers reasonable value compared with simpler inputs (Table *1*).

Tier and inputs	Potential clinical contribution	AI-specific opportunity	Practical limitation
Baseline: age, smoking, blood pressure, diabetes history	First-line risk assessment; feasible in many clinics	Check completeness; compare with locally appropriate validated scores	Measurement quality and missing data
Core laboratory: lipid profile, glucose or HbA1c, creatinine/eGFR	Improve risk classification and medication safety	Combine verified data with clinical variables	Access, assay quality, repeated-test costs
Selected risk enhancers: lipoprotein(a), hs-CRP, albuminuria	Refine decisions for selected patients	Evaluate incremental value only after local validation	Selective indications; variable availability
High-dimensional assays: proteomics, lipidomics, genomics	Research phenotyping and potential precision approaches	Discover patterns and test multimodal models	High cost, transportability, assay harmonization
Opportunistic images: ECG or chest radiograph	Possible flags for additional evaluation	Validated image-based risk or abnormality detection	Not interchangeable with confirmatory diagnosis

TABLE 1: Biomarker and data tiers for cardiovascular risk assessment in resource-limited settings

This table is a proposed service-design framework, not a guideline or an externally validated clinical score.

AI: artificial intelligence; ECG: electrocardiogram; eGFR: estimated glomerular filtration rate; HbA1c: hemoglobin A1c; hs-CRP: high-sensitivity C-reactive protein

Pharmacology perspective: turning risk estimates into safe treatment

A useful risk estimate should lead to safe and sustainable preventive treatment. In principle, AI could identify patients whose records suggest missed lipid-lowering therapy, missed monitoring, potential interactions, or poor adherence. Such alerts are aids to review, not prescriptions. Clinicians still need to consider indication, current guidelines, contraindications, patient preference, renal function, and drug availability. The 2026 multi-society dyslipidemia guideline supplies the clinical framework for contemporary lipid management; it should not be interpreted as evidence for AI-directed drug treatment [26].

Pharmacogenomic guidance is discussed here because the pharmacology step is where a risk estimate becomes a prescription, and any AI tool that suggests or reviews preventive drugs must stay consistent with established gene-drug recommendations. The guidance itself is not AI-derived. Clinical Pharmacogenetics Implementation Consortium (CPIC) recommendations for statins distinguish among specific drugs and transporter or metabolism phenotypes involving SLCO1B1, ABCG2, and CYP2C9 [27]. Other CPIC guidance addresses CYP2C19-related variation in clopidogrel activation, principally in clinical settings such as acute coronary syndrome or percutaneous coronary intervention, not routine primary prevention [28]. Table *2* summarizes selected adult scenarios; combined SLCO1B1 and ABCG2 findings may also affect recommended rosuvastatin dosing.

Gene	Drug(s) affected	Phenotype-dependent effect	Selected CPIC-informed action (adults)
SLCO1B1	Simvastatin; effects also vary across other statins	Decreased or poor transporter function increases simvastatin acid exposure and myopathy risk [27].	Decreased/possible decreased function: prefer another statin; if simvastatin is used, limit to <20 mg/day. Poor function: use an alternative statin [27].
ABCG2	Rosuvastatin	Poor function increases rosuvastatin exposure; myopathy risk is uncertain [27].	Poor function: start at ≤20 mg/day; consider another statin or combination if >20 mg is needed. Decreased function alone does not mandate dose reduction [27].
CYP2C9	Fluvastatin	Intermediate and poor metabolizer phenotypes increase fluvastatin exposure [27].	Intermediate metabolizer: start at ≤40 mg/day; poor metabolizer: start at ≤20 mg/day, then adjust according to clinical response and guidance [27].
CYP2C19	Clopidogrel (prodrug)	Intermediate/poor metabolism reduces active-metabolite generation and platelet inhibition [28].	Use an alternative P2Y12 inhibitor when appropriate in guideline-covered indications (e.g., ACS/PCI), considering contraindications [28].

TABLE 2: Pharmacogenomic gene-drug pairs relevant to cardiovascular prevention

Selected adult recommendations depend on the reported gene-function phenotype, specific drug, and clinical indication; consult the full CPIC tables before prescribing. For rosuvastatin, combined SLCO1B1 and ABCG2 phenotypes may require a lower starting dose than either single-gene summary. These are pharmacogenetic recommendations, not AI-derived treatment effects.

AI: artificial intelligence; ACS: acute coronary syndrome; CPIC: Clinical Pharmacogenetics Implementation Consortium; PCI: percutaneous coronary intervention; P2Y12: platelet adenosine diphosphate receptor

AI has three potential, but largely unproven, roles at this step. First, decision-support software can match genotype results already stored in a patient's record against CPIC tables and alert the prescriber; such alerts are usually rule-based rather than machine learning, but they form the infrastructure into which AI would be built. Second, machine-learning models can combine genotype with clinical variables such as age, renal function, co-medication, and adherence to predict drug response or adverse effects. These approaches have been studied mainly for warfarin dosing and in psychiatry and oncology [29], and cardiovascular applications, including polygenic models of drug response, remain at an early stage [30]. Third, natural-language processing could retrieve genotype results or records of previous drug intolerance from unstructured clinical notes. None of these applications has been shown to improve outcomes in primary prevention, and routine genotyping is rarely available in resource-limited services. In those settings, pharmacogenomics is therefore relevant mainly for patients who already have results, for example, after a coronary intervention, and as a constraint that any future AI prescribing tool must respect.

Even accurate treatment alerts have little value when the recommended medicine is unavailable or the patient cannot afford repeat visits. Automation may also increase workload if every alert produces an additional investigation without improving decisions. For these reasons, prescribing remains a clinician-controlled step in the proposed pathway.

Community medicine perspective: screening, linkage, and continuity

Community programs make clear what must happen after a risk estimate is generated: an individual must be reached, measured reliably, linked to a prescriber, and retained in care. Two non-AI trials offer useful comparisons. In PolyIran, a cluster-randomized trial involving 6,838 participants with and without prior CVD, major cardiovascular events occurred in 5.9% of the polypill group and 8.8% of the minimal care group (adjusted hazard ratio, 0.66; 95% confidence interval, 0.55-0.80) [31]. The overall benefit was demonstrated in a population comprising both primary and secondary prevention participants and should not be interpreted as an effect estimate restricted to primary prevention. HOPE 4 combined nonphysician health workers, physician-supervised medication, and family support; blood pressure control was 69% in the intervention group and 30% in usual care [32]. Neither result can be attributed to AI.

In SMARThealth India, community health workers used tablets to assess risk and facilitate referral and clinician decision support. Despite substantial delivery of the intervention, the stepped-wedge trial did not demonstrate improved blood pressure control [8]. Changes in measured blood pressure led to discordant risk categories, and only about one-third of the evaluated cohort was effectively exposed to the intervention. This finding is a warning for AI implementation: an unreliable measurement and referral chain can defeat even a technically capable digital tool.

Evidence involving community health workers and mobile clinical support suggests that task sharing is

feasible, while effectiveness depends on how consistently the program operates and whether patients complete care [33,34]. For a possible AI-enabled program, outputs should be understandable to frontline staff, usable with intermittent connectivity where necessary, and paired with explicit clinician escalation rules. The proposed tasks and oversight points in Table 3 translate these requirements into testable service components.

Pathway step	AI-supported task	Inputs	Human checkpoint	Evaluation measure
Community screening	Flag missing or implausible risk-factor entries; prompt repeat BP measurement	Health worker records, BP readings	Health worker repeats measurement	Missingness; repeat-measurement agreement
Laboratory testing	Prioritize patients for targeted tests; check result plausibility	Clinical risk tier, previous results	Laboratory and clinician review	Test yield; cost per reclassified patient
Risk estimation	Locally validated risk estimate	Verified clinical and laboratory variables	Clinician confirms risk category	Calibration; net benefit
Prescribing	Alerts for untreated eligible patients, interactions, renal dosing, and monitoring gaps	Prescriptions, eGFR, comorbidities	Prescriber accepts or overrides with reason	Appropriate initiation; override rate; adverse effects
Follow-up	Predict missed visits or nonadherence; send reminders in local languages	Dispensing and attendance records	Health worker contact and escalation	Persistence; follow-up retention

TABLE 3: Candidate AI-supported tasks across the cardiovascular prevention pathway

Proposed tasks for prospective evaluation; none should operate without the listed human checkpoint.

AI: artificial intelligence; BP: blood pressure; eGFR: estimated glomerular filtration rate

Conversational systems may assist with reminders and basic medication education, but adherence findings require careful qualification. A 2026 review of chatbot interventions across noncommunicable diseases included seven studies and reported improved adherence alongside very substantial heterogeneity [35]. Its mixed patient populations and outcome measures do not establish that chatbots prevent cardiovascular events in underserved communities. Completion of referrals, treatment uptake, persistence, and control of risk factors are more informative measures than numbers of messages delivered.

Randomized evidence: predictive accuracy is not clinical effectiveness

Randomized evidence should be interpreted according to the setting and endpoint actually studied. In EAGLE, 22,641 adults received routine electrocardiography, and access to an AI-based analysis increased the proportion with a newly identified low ejection fraction from 1.6% to 2.1% [36]. This supports diagnostic case finding, not a demonstrated reduction in first cardiovascular events. In a separate trial among hospitalized patients, mortality at 90 days was 3.6% with AI electrocardiogram alerts compared with 4.3% without them [37]. That result is clinically relevant but comes from acute hospital care rather than a community prevention program.

The broader trial literature offers an uneven picture. A 2026 systematic review of 31 randomized cardiovascular AI studies reported favorable findings for several intermediate outcomes. Its pooled analyses suggested fewer major adverse cardiovascular events with image-based decision support and a modest systolic blood pressure reduction with AI-enabled mobile health, but certainty was rated low or very low across outcomes [15]. More diagnoses or referrals are not necessarily improvements if they produce false positives, strain available services, or fail to lead to treatment. Studies should therefore distinguish algorithm performance, process changes, and patient-important outcomes.

Table 4 brings together the key studies discussed in this review. They were selected to represent each stage of evidence, from model development to clinical outcome trials, rather than to provide an exhaustive list. Part A contains seven studies that directly tested AI: two large development studies that compared machine-learning and conventional risk scores in primary prevention populations [12,13]; the most recent meta-analysis of such models [14]; one model that derives risk from data already collected when calculator inputs are missing [9]; two pragmatic randomized trials of AI tools in routine care, one with a diagnostic endpoint and one with mortality [36,37]; and the most recent meta-analysis of randomized trials of cardiovascular AI [15]. Part B contains four non-AI trials, included deliberately as benchmarks, because an AI-supported program is worth adopting only if it improves on what conventional strategies already achieve [18]. JUPITER shows what biomarker-guided selection for statin therapy can achieve without AI [23].

PolyIran and HOPE 4 show the event reduction and risk-factor control that simplified drug delivery and task-shared community care can achieve in middle-income settings [31,32]. SMARThealth India shows how a digital, non-AI decision-support program can fail through measurement and exposure gaps [8]. These four trials therefore define the comparator and the delivery conditions against which any AI tool for resource-limited prevention should be judged. None of the studies in Part A tested AI against a first-event clinical endpoint in a resource-limited setting.

Study and design	Setting	Key observation	Endpoint level	Reason for inclusion and interpretation
Part A. Studies that directly tested AI (n = 7)				
Weng et al. (2017); cohort [12]	UK primary care; high-income	378,256 patients; best ML AUC 0.764 versus 0.728 for conventional algorithm	Prediction (discrimination)	Large primary care comparison of ML with a conventional score; modest prediction gain, not a treatment-outcome study
Kakadiaris et al. (2018); cohort [13]	US multi-ethnic cohort with European validation; high-income	6,459 participants; ML calculator AUC 0.92 versus 0.71	Prediction (discrimination)	Head-to-head comparison with a guideline calculator; development-population result that needs local recalibration
Liu et al. (2025); meta-analysis [14]	Multiple countries	20 studies; pooled ML AUC up to 0.865 versus 0.765; I^2 >99%	Prediction (pooled discrimination)	Most recent synthesis of ML risk models; poor cross-study comparability and bias concerns
Weiss et al. (2024); risk prediction study [9]	US; high-income	Chest radiograph model; adjusted HR 1.73 for predicted risk ≥7.5% among 8,869 patients with missing inputs	Prognostic association (retrospective)	Uses data already collected when calculator inputs are missing; an opportunistic flag supported by retrospective evidence only
Yao et al. (2021); pragmatic RCT [36]	US; high-income	22,641 adults; new low-EF diagnosis 2.1% versus 1.6%	Diagnostic/process	Large pragmatic AI trial in routine primary care; diagnostic improvement, not first-event prevention
Lin et al. (2024); pragmatic RCT [37]	Taiwan, inpatients; high-income	15,965 inpatients; 90-day mortality 3.6% versus 4.3% (HR 0.83)	Clinical outcome (mortality)	One of few AI trials with a hard clinical endpoint; acute-care alerting, not transferable to community prevention
Ong et al. (2026); RCT meta-analysis [15]	Multiple countries	31 trials; image-based decision support MACE RR 0.74; mobile health SBP −3.18 mm Hg; low to very low certainty	Mixed (process, risk factor, clinical)	Most recent synthesis of AI trials; clinical-outcome evidence remains limited
Part B. Non-AI benchmark trials (n = 4)				
JUPITER (2008); RCT [23]	Multinational	17,802 participants with elevated hs-CRP; rosuvastatin HR 0.56 for primary end point	Clinical events	Benchmark for biomarker-guided preventive therapy achieved without AI
PolyIran (2019); cluster RCT [31]	Iran; middle-income	6,838 participants with and without prior CVD; major CVD events 5.9% versus 8.8% (adjusted HR 0.66; 95% CI 0.55–0.80)	Clinical events	Non-AI medication-delivery benchmark demonstrating fewer events in a mixed primary and secondary prevention population
HOPE 4 (2019); cluster RCT [32]	Colombia and Malaysia; middle-income	1,371 participants; BP control 69% versus 30%	Risk-factor control	Benchmark for task-shared community care in middle-income countries
SMARThealth India (2019); stepped-wedge	Rural India; middle-income	SBP target met 41.2% versus 39.2%; only 37.2% effectively exposed	Risk-factor control	Shows how a digital, non-AI program failed through measurement discordance and

RCT [8]				weak exposure

TABLE 4: Summary and appraisal of key evidence relevant to risk prediction, clinical action, and community delivery

Studies were selected to represent each stage of evidence, not as an exhaustive list or a quantitative meta-analysis. Part A includes studies that directly tested an AI method; Part B includes conventional, non-AI trials as benchmarks. The PolyIran overall result includes participants with and without prior CVD; no disputed subgroup estimate is reported.

AI: artificial intelligence; AUC: area under the receiver operating characteristic curve; BP: blood pressure; CVD: cardiovascular disease; EF: ejection fraction; HR: hazard ratio; hs-CRP: high-sensitivity C-reactive protein; I^2: between-study heterogeneity; MACE: major adverse cardiovascular events; ML: machine learning; RCT: randomized controlled trial; RR: risk ratio; SBP: systolic blood pressure

An integrated, resource-sensitive model

Figure 2 translates the multidisciplinary argument into a proposed clinical workflow. Frontline workers first record accessible risk factors such as age, smoking, diabetes, and blood pressure, using an appropriate local chart where indicated. Laboratory assessment is then targeted to the patient's needs and service capacity. AI may help identify incomplete records, prioritize follow-up, or apply a locally validated model, while clinicians confirm risk interpretation and all prescribing decisions. Repeat measurements and treatment responses feed back into subsequent community visits.

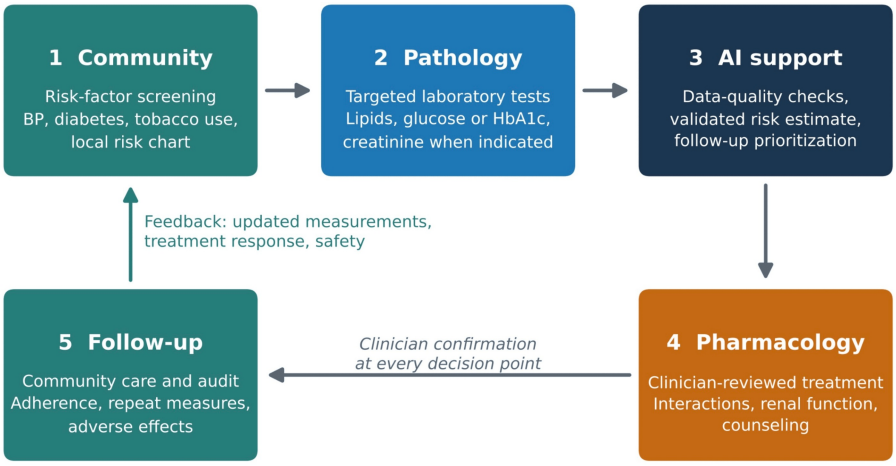


FIGURE 2: Proposed multidisciplinary pathway for AI-assisted cardiovascular prevention

Conceptual pathway integrating community assessment, targeted laboratory testing, clinician-reviewed treatment, and continued follow-up. AI may support data-quality checks, validated risk estimation, and prioritization but does not replace clinical confirmation or prescribing judgment. Arrows represent service flow, not proven causal effects. This is a framework for service design, not a validated clinical algorithm.

AI: artificial intelligence; BP: blood pressure; HbA1c: hemoglobin A1c

The figure is created using Canva (Canva Pty Ltd, Sydney, NSW, Australia); Image credit: Umer Farooq, Dimpal Modi, and Jeevan Rajguru

Before integrating such a tool into routine service, investigators should observe how it performs with real local data, ideally without allowing its predictions to influence care during the initial validation phase. A feasibility study could then examine whether staff can use the output safely. Pragmatic or cluster-randomized testing would be appropriate only once data quality, oversight, and safety procedures are established. Simpler models using consistently collected variables may provide better overall value than high-cost biomarker panels with only a small predictive advantage.

Validation, safety, equity, and implementation

Evaluation must extend beyond discrimination. TRIPOD+AI provides guidance for transparent reporting of prediction-model studies, whereas PROBAST+AI helps assess study quality, bias, and applicability [17,38]. For deployment decisions, investigators should describe missing-data handling, calibration level and slope, external performance, relevant subgroup differences, and what happens after algorithm updates. Decision-

curve or other clinical-utility analyses can then examine whether the risks of acting on the predictions are justified [39].

The potential harms of an AI-assisted pathway may be amplified where healthcare capacity is limited. Urban training datasets may poorly represent rural users; laboratory data may be absent for disadvantaged patients; variations in diagnoses and event recording can change model outputs; and automatic referrals may overwhelm receiving clinics. Data privacy also matters when community workers use shared devices. Implementation plans need restricted data access, accountable human review, referral escalation procedures, periodic audits, and predefined steps for disabling a model if concerning errors emerge (Table 5).

Risk	Safeguard	Indicator
Population shift and unequal coverage	External validation in rural and urban groups; periodic recalibration	Calibration by subgroup; screening reach
Poor-quality or missing input data	Repeat critical measurements; transparent missing-data handling	Missingness rate; repeat-test agreement
Unnecessary investigation or prescribing	Human review and action thresholds tied to guidelines	False-positive referrals; treatment appropriateness
Medication affordability and supply gaps	Link alerts to formulary and stock availability	Prescription fulfillment; persistence
Referral drop-off and weak follow-up	Community worker tracking and clear responsibility	Completed referrals; follow-up retention
Privacy and model drift	Restricted access, monitoring, periodic audits	Security incidents; change in model calibration
Overclaiming AI impact	Separate process, diagnostic, and clinical outcomes	Blood pressure, LDL-C, events, adverse effects

TABLE 5: Implementation risks, proposed safeguards, and measurable indicators

Proposed evaluation measures should be adapted to the local health system and the prospective study protocol.

AI: artificial intelligence; LDL-C: low-density lipoprotein cholesterol

Figure 3 sets out the sequence of evidence that should be in place before an AI tool is adopted in a prevention service. Its scientific basis is the established distinction between a prediction model's statistical performance and its effect on care [18,39], and each stage corresponds to an existing reporting standard: TRIPOD+AI and PROBAST+AI for model development and validation [17,38], DECIDE-AI for early clinical evaluation of AI decision support [40], and CONSORT-AI for randomized trials of AI interventions [41]. The four stages answer different questions and need different study designs. Stage 1 asks whether a model ranks and estimates risk accurately in the data used to build it. Stage 2 asks whether that performance holds in the intended population and its subgroups; this stage is especially important in resource-limited settings because transported models are often miscalibrated. Stage 3 asks whether staff can use the output safely and whether it changes clinical action in the intended direction without excess testing or workload. Stage 4 asks whether patients benefit, at what cost, and whether benefits are shared fairly. Success at one stage cannot be assumed to carry to the next: a higher AUC does not show that clinicians will act on the output, and appropriate clinical action does not show that events are prevented. Failure at any stage should lead to recalibration, redesign, or abandonment before further investment, and an intervention trial that finds no benefit is still useful when it reveals inconsistent measurements, incomplete referrals, or excess workload.

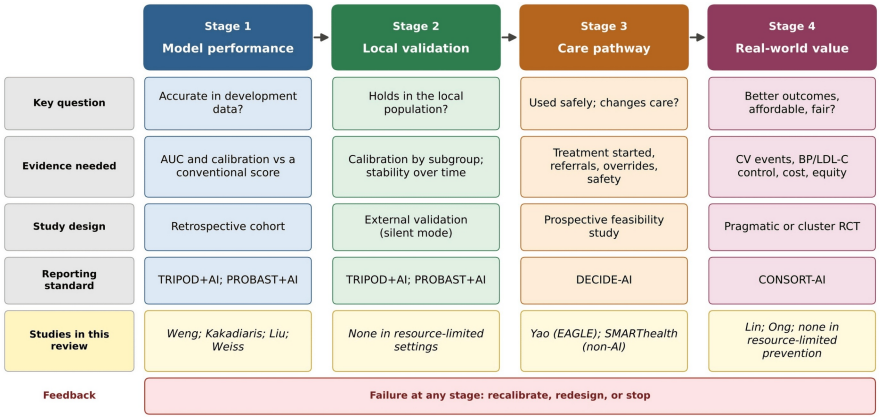


FIGURE 3: Evidence sequence for evaluating cardiovascular AI in resource-limited settings

Each column is a stage of evaluation; each row states what that stage must show and how. Stage-specific reporting standards are TRIPOD+AI and PROBAST+AI (Stages 1 and 2), DECIDE-AI (Stage 3), and CONSORT-AI (Stage 4). The bottom row maps the studies summarized in Table 4 onto the sequence. Success at an earlier stage does not establish benefit at the next stage; a higher area under the curve does not by itself demonstrate fewer cardiovascular events. Studies in this review [8,9,12-15,36,37].

AI: artificial intelligence; AUC: area under the receiver operating characteristic curve; BP: blood pressure; CONSORT-AI: Consolidated Standards of Reporting Trials-Artificial Intelligence; CV: cardiovascular; DECIDE-AI: Developmental and Exploratory Clinical Investigations of Decision support systems driven by Artificial Intelligence; LDL-C: low-density lipoprotein cholesterol; PROBAST+AI: Prediction model Risk Of Bias ASsessment Tool updated for AI; RCT: randomized controlled trial; TRIPOD+AI: Transparent Reporting of a multivariable prediction model for Individual Prognosis Or Diagnosis updated for AI

The figure is created using Canva (Canva Pty Ltd, Sydney, NSW, Australia); Image credit: Dimpal Modi and Bipin Chavda

Workforce readiness and interdisciplinary implementation

A technically sound model can still fail if staff lack the support needed to use it. Joseph et al. reviewed AI across clinical care and medical education, including cardiac monitoring, and discussed familiarity with the technology, privacy, accountability, cost, and adoption barriers [42]; as a broad review rather than a prevention trial, it frames workforce and governance questions but not treatment effectiveness. In an integrated program, laboratory personnel, prescribers, and community workers would need role-specific preparation: checking data accuracy, communicating absolute risk, recognizing false alarms, recording why alerts are accepted or overridden, and arranging timely referral. Future evaluations should assess whether staff make safe decisions as well as whether patients obtain medicines, attend follow-up, and receive equitable care.

Knowledge gaps and research priorities

The evidence identified in this review suggests that AI-supported interventions can improve selected cardiovascular risk factors and aspects of clinical care. However, evidence demonstrating a reduction in first cardiovascular events through AI-supported prevention programs in resource-limited primary care settings remains limited. For example, a secondary analysis of a randomized trial conducted in India evaluated an AI-enabled digital-twin intervention among 319 participants with type 2 diabetes. After one year, the intervention group showed greater reductions in systolic blood pressure than the standard care group (7.6 versus 3.2 mmHg), alongside improvements in selected hypertension-related outcomes and medication use [43]. Although relevant to cardiovascular risk-factor management in India, this study did not establish whether the intervention reduced first cardiovascular events or improved outcomes compared with a locally validated conventional cardiovascular risk assessment pathway.

Three important questions therefore remain. First, can affordable biomarkers improve treatment decisions sufficiently to justify their additional cost beyond locally validated risk scores? Second, can AI-supported care improve the initiation and long-term continuation of preventive medications without increasing unnecessary investigations or adverse effects? Third, can AI-assisted community programs improve screening, referral completion, and sustained follow-up among underserved populations? These questions should be evaluated against clearly defined conventional care pathways rather than against the absence of organized preventive services.

Future studies should distinguish improvements in care processes, such as screening uptake, completed

referrals, and medication adherence, from changes in cardiovascular risk factors, such as blood pressure and LDL-C levels, and from clinical outcomes, particularly first cardiovascular events. Trials with sufficient follow-up should assess whether improvements in intermediate outcomes translate into fewer cardiovascular events. Evaluations should also examine differences across age, sex, geographic location, socioeconomic groups, and data completeness to identify potential inequalities. Economic assessments should account for staff time, diagnostic tests, devices, connectivity, software maintenance, and subsequent healthcare visits. Collaboration among pathology, pharmacology, and community medicine services could help develop and evaluate interventions suited to resource-limited populations.

Limitations

This review is intentionally selective and has limitations. As a nonsystematic review of the literature, source selection was purposive rather than governed by predefined eligibility criteria, and no formal per-study risk-of-bias scoring or pooling was performed; selection bias cannot be excluded despite the safeguards described in the Review, and the conclusions should be read as an interpretive synthesis rather than a quantitative estimate of effect. Only PubMed was searched at the query level; relevant work indexed primarily in engineering, computer science, regional, or implementation databases may have been missed. Studies also used different populations, outcomes, prediction horizons, and comparators, making numerical pooling inappropriate. Several important community trials evaluated conventional digital or team-based care, not AI, and were discussed solely to illuminate delivery conditions. Finally, much of the published model-development evidence originates in health systems whose datasets and infrastructure differ from those of the resource-limited settings considered here.

Conclusions

AI may strengthen cardiovascular prevention when it helps existing services use dependable information and act on it consistently. The immediate priority in resource-limited settings is unlikely to be more complex prediction at any cost. Instead, locally tested tools should support affordable testing, guideline-based prescribing, safe follow-up, and timely referral without displacing clinical judgment. Pathology, pharmacology, and community medicine each contribute safeguards that an algorithm cannot supply by itself. Whether an AI-supported program deserves adoption must ultimately depend on local calibration, fair access, safety, cost, and demonstrable benefits for patients.

Appendices

Appendix 1

Search Strategy, Methodological Boundaries, and Bibliographic Evidence Matrix

Information sources and dates

PubMed (including MEDLINE-indexed records) was searched from database inception, with no lower publication-date limit, to October 31, 2026. Four initial thematic searches were performed on October 28, 2026; the strategy was refined to six searches and run on October 31, 2026. A publication-date upper limit of October 31, 2026 was applied to each refined search. No human, language, or study-design filters were imposed electronically. MeSH terms were combined with free-text terms restricted to titles and abstracts, where specified. Embase, Scopus, Web of Science, Google Scholar, and the Cochrane Library were not systematically searched, and the review does not claim otherwise.

The search was designed for a focused Review of the Literature, not a systematic review or PRISMA-based synthesis. AND linked distinct concepts, OR combined synonyms within concepts, and parentheses retained the intended search logic. Truncation (*) was used where appropriate to capture related word endings. The searches were intentionally broad; they overlapped, and not every returned record was screened. Purposive selection of relevant publications and backward reference checking complemented these queries. Counts below are database matches for each query, not unique records screened or studies included. They may change as PubMed indexing is updated.

ID	Search focus	Records
S1	AI and cardiovascular risk prediction	4,781
S2	Pathology: biomarkers and multimodal data	1,941
S3	Pharmacology: treatment and adherence	350
S4	Community medicine: primary care and screening	1,586
S5	External validation and equity	3,000
S6	Supporting non-AI implementation evidence	910

TABLE 6: Refined PubMed Boolean searches and retrieval results

S1. AI and cardiovascular risk prediction - 4,781 matching PubMed records

("Cardiovascular Diseases"[MeSH Terms] OR cardiovascular[Title/Abstract] OR hypertension[Title/Abstract]) AND ("Artificial Intelligence"[MeSH Terms] OR "Machine Learning"[MeSH Terms] OR "machine learning"[Title/Abstract] OR "deep learning"[Title/Abstract]) AND ("primary prevention"[Title/Abstract] OR "risk prediction"[Title/Abstract] OR "risk stratification"[Title/Abstract] OR "Risk Assessment"[MeSH Terms])

S2. Pathology: biomarkers and multimodal data - 1,941 matching PubMed records

(cardiovascular[Title/Abstract] OR atherosclerosis[Title/Abstract]) AND ("artificial intelligence"[Title/Abstract] OR "machine learning"[Title/Abstract] OR "deep learning"[Title/Abstract]) AND (biomarker*[Title/Abstract] OR proteomic*[Title/Abstract] OR lipidomic*[Title/Abstract] OR "lipoprotein(a)"[Title/Abstract] OR "C-reactive protein"[Title/Abstract] OR multiomic*[Title/Abstract])

S3. Pharmacology: treatment and adherence - 350 matching PubMed records

(cardiovascular[Title/Abstract] OR hypertension[Title/Abstract] OR dyslipidemia[Title/Abstract]) AND ("artificial intelligence"[Title/Abstract] OR "machine learning"[Title/Abstract] OR "clinical decision support"[Title/Abstract]) AND (statin*[Title/Abstract] OR pharmacotherap*[Title/Abstract] OR pharmacogenom*[Title/Abstract] OR "drug therapy"[Title/Abstract] OR "medication adherence"[Title/Abstract])

S4. Community medicine: primary care and screening - 1,586 matching PubMed records

(cardiovascular[Title/Abstract] OR hypertension[Title/Abstract]) AND ("artificial intelligence"[Title/Abstract] OR "machine learning"[Title/Abstract] OR "AI-assisted"[Title/Abstract]) AND ("primary care"[Title/Abstract] OR "community health"[Title/Abstract] OR rural[Title/Abstract] OR "low- and middle-income"[Title/Abstract] OR "resource-limited"[Title/Abstract] OR screening[Title/Abstract])

S5. External validation and equity - 3,000 matching PubMed records

(cardiovascular[Title/Abstract] OR "risk prediction"[Title/Abstract]) AND ("artificial intelligence"[Title/Abstract] OR "machine learning"[Title/Abstract]) AND ("external validation"[Title/Abstract] OR calibration[Title/Abstract] OR transportability[Title/Abstract] OR "clinical utility"[Title/Abstract] OR fairness[Title/Abstract] OR equity[Title/Abstract])

S6. Supporting non-AI implementation evidence - 910 matching PubMed records

(cardiovascular[Title/Abstract] OR hypertension[Title/Abstract]) AND ("community health workers"[Title/Abstract] OR "mobile health"[Title/Abstract] OR "clinical decision support"[Title/Abstract] OR "fixed-dose combination"[Title/Abstract]) AND (prevention[Title/Abstract] OR "risk management"[Title/Abstract] OR "blood pressure control"[Title/Abstract])

Retrieval date: October 31, 2026, for S1-S6 (publication-date filter through October 31, 2026). Total counts must not be summed as unique records because queries overlap. The S6 search is a contextual search for care-delivery evidence and does not establish AI effectiveness. No PRISMA flow diagram, deduplication count, or exhaustive screening total is claimed.

Appendix 2

SANRA item	How it is addressed in this review	Section
1. Importance of the topic	Burden of cardiovascular disease, specific barriers to conventional risk prediction in resource-limited settings, and the gap in AI evidence	Introduction & Background
2. Aims of the review	Three numbered objectives	Introduction & Background
3. Literature search	Database, date range, run dates, Boolean strings, and retrieval counts; explicit selection criteria	Review design and rationale; Literature search; Appendix 1
4. Referencing	Key statements referenced; bibliographic details and DOIs checked against PubMed	Throughout; References
5. Presentation of level of evidence	Each key study labeled by design, setting, whether AI was directly tested, and endpoint level	Table 4; Randomized evidence section
6. Presentation of relevant endpoint data	Effect sizes reported for key studies, with certainty ratings from source syntheses where available	Tables 2 and 4; Randomized evidence section

TABLE 7: SANRA-based reporting checklist

SANRA items are summarized from Baethge et al. [16]; the scale is intended for nonsystematic review articles.

DOI: Digital Object Identifier; SANRA: Scale for the Assessment of Narrative Review Articles

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Disclosures

AI use statement: The authors used Claude Sonnet 5.5 (Anthropic, PBC, San Francisco, CA, USA) as follows: language polishing, structure or formatting in Methodology. Claude Sonnet 5.5 was used for formatting only and not generating or comparing any data. The authors confirm that they have reviewed and verified all AI-assisted content and take full responsibility for the work. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Associate Editor authorship:** Neena Bhatti was an Associate Editor for the Cureus Journal of Medical Science at the time of submission. To minimize potential bias, this manuscript was handled independently by an alternate Associate Editor with no conflict of interest, and the author was blinded to and had no access to the peer review process, reviewer identities, or editorial decision-making for this submission. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

References

1. GBD 2023 Causes of Death Collaborators: Global burden of 292 causes of death in 204 countries and territories and 660 subnational locations, 1990-2023: a systematic analysis for the Global Burden of Disease Study 2023. Lancet. 2025, 406:1811-72. 10.1016/S0140-6736(25)01917-8

2. World Health Organization cardiovascular disease risk charts: revised models to estimate risk in 21 global regions. Lancet Glob Health. 2019, 7:e1332-45. 10.1016/S2214-109X(19)30318-3
3. Fleming KA, Horton S, Wilson ML, et al.: The Lancet Commission on diagnostics: transforming access to diagnostics. Lancet. 2021, 398:1997-2050. 10.1016/S0140-6736(21)00673-5
4. Birhanu MM, Evans RG, Zengin A, et al.: Absolute cardiovascular risk scores and medication use in rural India: a cross-sectional study. BMJ Open. 2022, 12:e054617. 10.1136/bmjopen-2021-054617
5. Birhanu MM, Zengin A, Evans RG, et al.: Comparison of laboratory-based and non-laboratory-based cardiovascular risk prediction tools in rural India. Trop Med Int Health. 2025, 30:57-64. 10.1111/tmi.14069
6. Marcus ME, Manne-Goehler J, Theilmann M, et al.: Use of statins for the prevention of cardiovascular disease in 41 low-income and middle-income countries: a cross-sectional study of nationally representative, individual-level data. Lancet Glob Health. 2022, 10:e369-79. 10.1016/S2214-109X(21)00551-9
7. Geldsetzer P, Manne-Goehler J, Marcus ME, et al.: The state of hypertension care in 44 low-income and middle-income countries: a cross-sectional study of nationally representative individual-level data from 1.1 million adults. Lancet. 2019, 394:652-62. 10.1016/S0140-6736(19)30955-9
8. Peiris D, Praveen D, Mogulluru K, et al.: SMARThealth India: a stepped-wedge, cluster randomised controlled trial of a community health worker managed mobile health intervention for people assessed at high cardiovascular disease risk in rural India. PLoS One. 2019, 14:e0213708. 10.1371/journal.pone.0213708
9. Weiss J, Raghu VK, Paruchuri K, Zinzuwadia A, Natarajan P, Aerts HJ, Lu MT: Deep learning to estimate cardiovascular risk from chest radiographs: a risk prediction study. Ann Intern Med. 2024, 177:409-17. 10.7326/M23-1898
10. Khan SS, Matsushita K, Sang Y, et al.: Development and validation of the American Heart Association's PREVENT equations. Circulation. 2024, 149:430-49. 10.1161/CIRCULATIONAHA.123.067626
11. Armoundas AA, Narayan SM, Arnett DK, et al.: Use of artificial intelligence in improving outcomes in heart disease: a scientific statement from the American Heart Association. Circulation. 2024, 149:e1028-50. 10.1161/CIR.0000000000001201
12. Weng SF, Reps J, Kai J, Garibaldi JM, Qureshi N: Can machine-learning improve cardiovascular risk prediction using routine clinical data?. PLoS One. 2017, 12:e0174944. 10.1371/journal.pone.0174944
13. Kakadiaris IA, Vrigkas M, Yen AA, Kuznetsova T, Budoff M, Naghavi M: Machine learning outperforms ACC/AHA CVD risk calculator in MESA. J Am Heart Assoc. 2018, 7:e009476. 10.1161/JAHA.118.009476
14. Liu T, Krentz A, Lu L, Curcin V: Machine learning based prediction models for cardiovascular disease risk using electronic health records data: systematic review and meta-analysis. Eur Heart J Digit Health. 2025, 6:7-22. 10.1093/ehjdh/ztae080
15. Ong AQ, Ang CS, Bojic I, et al.: Artificial intelligence in cardiovascular care: a systematic review and meta-analysis of randomised controlled trials. EClinicalMedicine. 2026, 98:104123. 10.1016/j.eclinm.2026.104123
16. Baethge C, Goldbeck-Wood S, Mertens S: SANRA-a scale for the quality assessment of narrative review articles. Res Integr Peer Rev. 2019, 4:5. 10.1186/s41073-019-0064-8
17. Moons KG, Damen JA, Kaul T, et al.: PROBAST+AI: an updated quality, risk of bias, and applicability assessment tool for prediction models using regression or artificial intelligence methods. BMJ. 2025, 388:e082505. 10.1136/bmj-2024-082505
18. Khan SS, Greenland P, Hayman LL, et al.: Criteria to assess the predictive and clinical utility of novel models, biomarkers, and tools for risk of cardiovascular disease: a scientific statement from the American Heart Association. Circulation. 2026, 153:e953-70. 10.1161/CIR.0000000000001401
19. Scheuermann B, Brown A, Colburn T, Hakeem H, Chow CH, Ade C: External validation of the American Heart Association PREVENT cardiovascular disease risk equations. JAMA Netw Open. 2024, 7:e2438311. 10.1001/jamanetworkopen.2024.38311
20. Libby P: Inflammation and the pathogenesis of atherosclerosis . Vascul Pharmacol. 2024, 154:107255. 10.1016/j.vph.2023.107255
21. Reyes-Soffer G, Yeang C, Michos ED, Boatwright W, Ballantyne CM: High lipoprotein(a): actionable strategies for risk assessment and mitigation. Am J Prev Cardiol. 2024, 18:100651. 10.1016/j.ajpc.2024.100651
22. Nurmohamed NS, Kraaijenhof JM, Mayr M, Nicholls SJ, Koenig W, Catapano AL, Stroes ES: Proteomics and lipidomics in atherosclerotic cardiovascular disease risk prediction. Eur Heart J. 2023, 44:1594-607. 10.1093/eurheartj/ehad161
23. Ridker PM, Danielson E, Fonseca FA, et al.: Rosuvastatin to prevent vascular events in men and women with elevated C-reactive protein. N Engl J Med. 2008, 359:2195-207. 10.1056/NEJMoa0807646
24. Ridker PM, Everett BM, Thuren T, et al.: Antiinflammatory therapy with canakinumab for atherosclerotic disease. N Engl J Med. 2017, 377:1119-31. 10.1056/NEJMoa1707914
25. Nordestgaard LT, Wolford BN, de Gonzalo-Calvo D, et al.: Multiomics in atherosclerotic cardiovascular disease. Atherosclerosis. 2025, 408:120414. 10.1016/j.atherosclerosis.2025.120414
26. Blumenthal RS, Morris PB, Gaudino M, et al.: 2026 ACC/AHA/AACVPR/ABC/ACPM/ADA/AGS/APhA/ASPC/NLA/PCNA guideline on the management of dyslipidemia: a report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. J Am Coll Cardiol. 2026, 87:2624-757. 10.1016/j.jacc.2025.11.016
27. Cooper-DeHoff RM, Niemi M, Ramsey LB, et al.: The Clinical Pharmacogenetics Implementation Consortium guideline for SLCO1B1, ABCG2, and CYP2C9 genotypes and statin-associated musculoskeletal symptoms. Clin Pharmacol Ther. 2022, 111:1007-21. 10.1002/cpt.2557
28. Lee CR, Luzum JA, Sangkuhl K, et al.: Clinical Pharmacogenetics Implementation Consortium guideline for CYP2C19 genotype and clopidogrel therapy: 2022 update. Clin Pharmacol Ther. 2022, 112:959-67. 10.1002/cpt.2526
29. Cilluffo G, Fasola S, Ferrante G, Malizia V, Montalbano L, La Grutta S: Machine learning: an overview and applications in pharmacogenetics. Genes (Basel). 2021, 12:1511. 10.3390/genes12101511
30. Ramayanam NR, Bukke SP, Thalluri C, et al.: Combinations of single gene predictors with AI-driven polygenic precision therapies in cardiovascular diseases. Pharmacogenomics. 2026, 27:279-86. 10.1080/14622416.2026.2686336

31. Roshandel G, Khoshnia M, Poustchi H, et al.: Effectiveness of polypill for primary and secondary prevention of cardiovascular diseases (PolyIran): a pragmatic, cluster-randomised trial. Lancet. 2019, 394:672-83. 10.1016/S0140-6736(19)31791-X
32. Schwalm JD, McCready T, Lopez-Jaramillo P, et al.: A community-based comprehensive intervention to reduce cardiovascular risk in hypertension (HOPE 4): a cluster-randomised controlled trial. Lancet. 2019, 394:1231-42. 10.1016/S0140-6736(19)31949-X
33. Jeet G, Thakur JS, Prinja S, Singh M: Community health workers for non-communicable diseases prevention and control in developing countries: evidence and implications. PLoS One. 2017, 12:e0180640. 10.1371/journal.pone.0180640
34. Agarwal S, Glenton C, Tamrat T, et al.: Decision-support tools via mobile devices to improve quality of care in primary healthcare settings. Cochrane Database Syst Rev. 2021, 7:CD012944. 10.1002/14651858.CD012944.pub2
35. Chen S, Fang Y, Ding L, Mo PK, Wang Z: Artificial intelligence-based chatbots to enhance medication adherence among patients with non-communicable chronic diseases: systematic review and meta-analysis. PLOS Digit Health. 2026, 5:e0001507. 10.1371/journal.pdig.0001507
36. Yao X, Rushlow DR, Inselman JW, et al.: Artificial intelligence-enabled electrocardiograms for identification of patients with low ejection fraction: a pragmatic, randomized clinical trial. Nat Med. 2021, 27:815-9. 10.1038/s41591-021-01335-4
37. Lin CS, Liu WT, Tsai DJ, et al.: AI-enabled electrocardiography alert intervention and all-cause mortality: a pragmatic randomized clinical trial. Nat Med. 2024, 30:1461-70. 10.1038/s41591-024-02961-4
38. Collins GS, Moons KG, Dhiman P, et al.: TRIPOD+AI statement: updated guidance for reporting clinical prediction models that use regression or machine learning methods. BMJ. 2024, 385:e078378. 10.1136/bmj-2023-078378
39. Steyerberg EW, Vickers AJ, Cook NR, et al.: Assessing the performance of prediction models: a framework for traditional and novel measures. Epidemiology. 2010, 21:128-38. 10.1097/EDE.0b013e3181c30fb2
40. Vasey B, Nagendran M, Campbell B, et al.: Reporting guideline for the early stage clinical evaluation of decision support systems driven by artificial intelligence: DECIDE-AI. BMJ. 2022, 377:e070904. 10.1136/bmj-2022-070904
41. Liu X, Cruz Rivera S, Moher D, Calvert MJ, Denniston AK: Reporting guidelines for clinical trial reports for interventions involving artificial intelligence: the CONSORT-AI extension. Nat Med. 2020, 26:1364-74. 10.1038/s41591-020-1034-x
42. Joseph G, Bhatti N, Mittal R, Bhatti A: Current application and future prospects of artificial intelligence in healthcare and medical education: a review of literature. Cureus. 2025, 17:e77313. 10.7759/cureus.77313
43. Shamanna P, Joshi S, Dharmalingam M, et al.: Digital twin in managing hypertension among people with type 2 diabetes: 1-year randomized controlled trial. JACC Adv. 2024, 3:101172. 10.1016/j.jacadv.2024.101172